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Diagnosis and management of idiopathic omental infarction: A case report

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ABSTRACT

A 32 year old man presented to casualty on three occasions in the space of four days, with intermittent, worsening abdominal pain. These symptoms imitated other commoner causes of acute abdomen and the site of onset changed, both factors delaying diagnosis. In due course, computer tomography imaging established findings indicative of omental infarction. Patient was discharged from hospital nine days later, having made a satisfactory recovery following successful conservative treatment. In this report, we evaluate the merits of a similar approach in future instances.

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1. Introduction

Idiopathic omental infarction (OI), though uncommon, is increasingly being reported in general surgical and radiological literature, on account of advancing imaging techniques and improved recognition of its radiographic presentation.[1,2] The omentum is a fat laden peritoneal remnant of embryological development and anatomically divided into the greater and lesser omentum. Multiple aetiological factors have been associated with OI, rendering infarcted omental tissue. Here we report a case of idiopathic OI, drawing particular attention to contemporary diagnostic and management considerations.

2. Case report

A 32 year old office worker previously fit and well, experienced a sudden onset, cramping abdominal pain, resembling 'a stitch'. It localised lateral to the umbilicus on the right side, and radiated to the lumbar and flank regions. Escalating in intensity through the day, simple analgesia having minimal effect, he presented to casualty in the evening, requiring tramadol hydrochloride to ease the pain. Movement seemed to aggravate it. An atypical appendicitis or renal colic was primarily queried, but neither was a convincing fit with the clinical presentation. There was no past surgical or medical history. Urine analysis and inflammatory markers were both normal. Requested plain film abdominal and erect chest radiographs showed no active disease. The pain gradually settled and the patient was discharged with a diagnosis of non specific abdominal pain; a short course of mild opiates was dispensed.

A second admission the following day revealed much the same in presentation and blood investigations, the symptoms eventually resolving after 2–3 h observation.

Seen by the authors on his third hospital admission in four days, the deteriorating, intermittent pain was now severe enough to require intramuscular administration of morphine sulphate. Much as before, the pain had a cramp likened character, now with epigastrium localisation and radiation to both right upper and lower quadrants. Examination revealed rebound tenderness in the epigastrium and a soft, diffusely tender abdomen otherwise. Hernial orifices were clear and rectal examination was normal. But for a mildly raised C-reactive protein (18 mg/L), blood investigations were all within normal range, and no comparative change was seen in imaging studies from four days prior.

The apparent dissociation between pain severity and clinical findings puzzled us. To ensure gallstone disease went unmissed, ultrasonography was requested in the first instance. Concern however grew about a possible organ perforation, as the patient developed a clinically guarding abdomen. An urgent computed tomography scan performed illustrated increased density and stranding in omental fat in the right upper and lower quadrant, suggestive of segmental infarction of omentum (Figs. 1 and 2).

The patient was treated conservatively with analgesia, anti-inflammatory medication and fluid management, coupled with daily monitoring of blood inflammatory markers. An increase in these markers and/or unresolved pain will have indicated surgical intervention. As it happens, his pain resolved and the patient was discharged without complication, a total of nine days from onset of initial symptoms.

3. Discussion

Omental infarction is a rare cause of acute abdomen, with an incidence equivalent to less than four cases per 1000 cases of appendicitis.[3] Low incidence and non specific presentation contribute to OI being misconstrued for appendicitis, peptic ulcer disease, cholecystitis, pancreatitis, among other abdominal pathology.[2–6] Sequelae – morbidity and mortality – potentially inflicted by these diagnoses, offset by a comparatively innocuous

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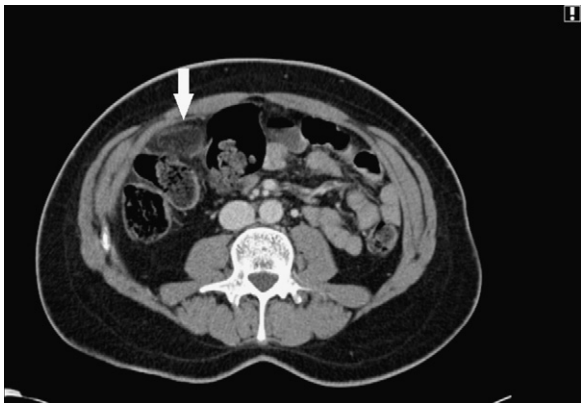

Fig. 1. Axial plane. Note change in omental tissue on right side of abdomen (white arrow).

recovery associated with OI, rationalised mandatory exploratory surgery in earlier reportings.[7] More recent literature questions the indication for surgery, as improvements in accuracy and accessibility of CT imaging diagnosis have been made.[3]

Infarction can be an isolated event, or in succession to omental torsion. Bush is credited with citing the very first case of omental infarction associated, with haemorrhaging into the greater omentum caused by a traumatic event,[7] and Eitel, the first case of torsion associated OI, a few years later in 1896.[8] Both phenomena can be further classed as primary and secondary depending on pathogenesis.[3] A diagnosis of primary or 'idiopathic' OI is made when no discernable aetiology is found. Secondary causes for OI include hypercoagulability, vasculitides, polycythaemia, and for omental torsion, cysts, tumours, and adhesions. Primary causes, or contributing factors, to omental torsion encompass obesity, local trauma, heavy food intake, coughing, sudden body movements, laxative use and hyperperistalsis.[2]

Pathogenesis relating to blood supply disruption in idiopathic OI is unknown. In view of a preponderance for right side presentations,[4,10] it has been suggested that the right half of the omentum consists of anatomically altered vasculature, less tolerant of spontaneous venous stasis and thrombosis secondary to stretching of omental veins.[5,11] Interestingly a raised body mass index has been of particular interest, on the back of cases reporting idiopathic OI in obese children. It is hypothesised that fatty accumulation in the omentum impedes the distal right epiploic artery, and additional structural mass potentially precipitates torsion.[12]

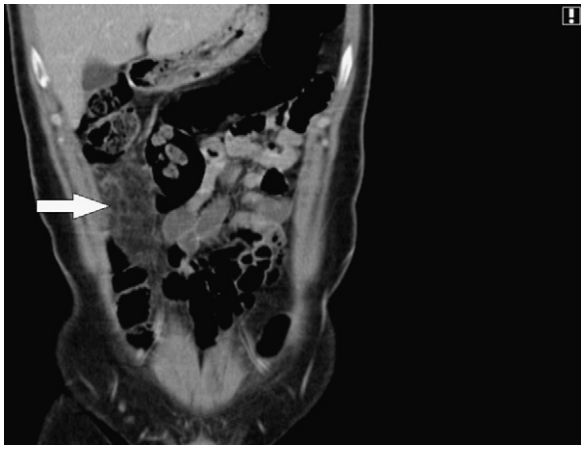

Fig. 2. Coronal plane.

Progression of torsion to OI is more straightforward to explain. Redundant omentum twisting on itself at a fulcrum customarily causes vascular kinking in the structure at the level of contortion, in turn causing venous stasis and thrombosis at this point.[2] A longer, mobile right side greater omentum will therefore potentially inflict this part to more torsion induced infarction.[1]

Understanding abdominal innervation helps to decipher clinical presentation. Parietal peritoneum of the anterior abdominal wall shares somatic innervations with muscle and skin adjacently overlying. Irritation will therefore precipitate localised tenderness and muscle contraction, guarding, through efferent pathways, via afferent connections on the parietal peritoneum. Owing to its surface area and proximity to the anterior wall, and coupled with extensive manoeuvrability, diseased greater omentum can irritate parietal peritoneum in numerous locations across almost the entire anterior abdomen: which is why greater omentum is a strong candidate for causing multi foci, site specified pain, mirroring our patient's presentation.[9]

Expanded access to CT imaging over the last twenty years has coincided with improved recognition of omental pathology radiographically.[13] Localised fat density lesions are seen in OI.[10,13–15] Concentric linear strands, or the 'whirl' sign,[3,13] and hyperattentuated streaky infiltration[1] have both been described in omental torsion. Definitive differentiation between OI and torsion related infarction can only be made surgically, although discerning between them does not change management.

There is, at present, no authoritative course of action for managing omental infarction. Accurate recognition of omental pathology on CT imaging means diagnosis can be attained without exploratory surgery, and in turn conservative management has become a viable option. This approach utilises analgesics and anti-inflammatory medication with optimal fluid management in the first instance.[1] General consensus holds OI and omental torsion as principally self limiting conditions, and this is supported by CT imaging data at 1–3 years follow up.[14] In tandem there are general anaesthetic and surgical risks to consider with any surgical intervention. Park et al.[1] argue that, on collective balance, surgery should not be first line of management – particularly as better imaging accessibility forgoes the need for investigative surgery. Reviewing 14 cases diagnosed with imaging studies, 11 were successfully treated conservatively, with deteriorating symptoms necessitating surgery in the remainder.[1] This corroborates with data published on OI[10,16,17] and torsion associated OI[3,18] elsewhere. Fragoso et al. reason that the risks associated with conservative management are theoretical, with no reported cases to date.[12]

Of the authors recommending surgical management, a laparascopic approach is favoured, affording thorough abdominal exploration and omental necrosectomy.[2] The argument is, expediting surgery potentially hastens symptom resolution and enables patient discharge much sooner post operation.[5,6] Symptoms can otherwise persist for an average of 13.5 days through conservative management.[2] Further still, removing devitalised omentum reduces a 'theoretical' risk of secondary peritoneal abscess formation.[2,19,20]

As yet, no comparative study demonstrates a significant difference in outcome following surgery and conservative management. With opinions conflicting and the availability of robust evidence scant, decisions will invariably arise from collaborative experience. After CT imaging diagnosis, Itenburg et al. advocate close monitoring of a patient in the first 24–48 h, refraining from considering surgery until deterioration in any symptom, sign or clinical marker. To what extent a change is significant enough to precipitate surgery is an arbitrary judgement. Nevertheless prudence can potentially avoid unnecessary surgery, such as in our patient, circumventing its associated risks.[3]

4. Conclusion

We present a patient diagnosed with OI managed conservatively. Accounting for our own experiences and after scrutinising past literature, we propose, after radiographic diagnosis, an initial conservative approach is trialled. This should be dovetailed with frequent reassessment and regular monitoring of blood inflammatory markers, and a low threshold for pursuing surgical management.

Conflict of interest

None.

Funding

None.

Ethical approval

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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